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PRACTICAL PRESCRIBING

Practical Prescribing: Potassium sparing diuretics

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Practical Prescribing is a series produced in conjunction with the *Drug and Therapeutics Bulletin* to highlight important issues for prescribers to consider and prompts for shared decision making between prescribers, patients, and their carers. Targeted at all medical and non-medical prescribers, particularly doctors in training, the series covers medicines commonly prescribed in primary and secondary care.

Advisers to this series are Fraz Mir, consultant physician at Addenbrooke's Hospital, Cambridge, and David Phizackerley, former deputy editor of *Drug and Therapeutics Bulletin*.

What you need to know

- Potassium sparing diuretics are effective in treating resistant hypertension, and are useful for mild hypervolaemia, or for potentiating other diuretics
- Monitor potassium levels and renal function for the risk of hyperkalaemia
- The aldosterone inhibiting potassium sparing diuretics (spironolactone, eplerenone, finerenone) offer improvement in outcomes for patients with heart failure with reduced ejection fraction and heart failure with preserved ejection fraction, but also require monitoring
- Variations in subtype (aldosterone inhibiting *v* epithelial sodium channel blocker) might have an impact on drug selection

A 73 year old woman presents to her GP after checking her blood pressure at home. Her average reading over the past few months is 140/80 mmHg. She is taking a thiazide-type diuretic, a calcium channel blocker, and an angiotensin converting enzyme (ACE) inhibitor. Her ejection fraction is 60%, with diastolic dysfunction on echocardiogram. She has noted some mild lower extremity oedema. She has normal renal function and no other comorbidities of note. She exercises regularly and eats a Mediterranean-style diet. You consider offering a potassium sparing diuretic to treat her heart failure, hypertension, and mild oedema.

How often are potassium sparing diuretics prescribed and how do they work?

Potassium sparing diuretics are widely used in the management of hypertension, heart failure, and oedema. All potassium sparing diuretics increase sodium and water excretion while retaining potassium by blocking sodium channels in the distal portion of the nephron—specifically, the late distal tubule and collecting duct (fig 1). Compared with loop diuretics, they are considerably weaker (approximately 5% to 10% as potent), although urine output is dependent on dose.[1] Potassium sparing diuretics can be combined with other diuretics (such as torasemide, furosemide, and/or chlortalidone) to enhance fluid removal while preventing potassium loss.

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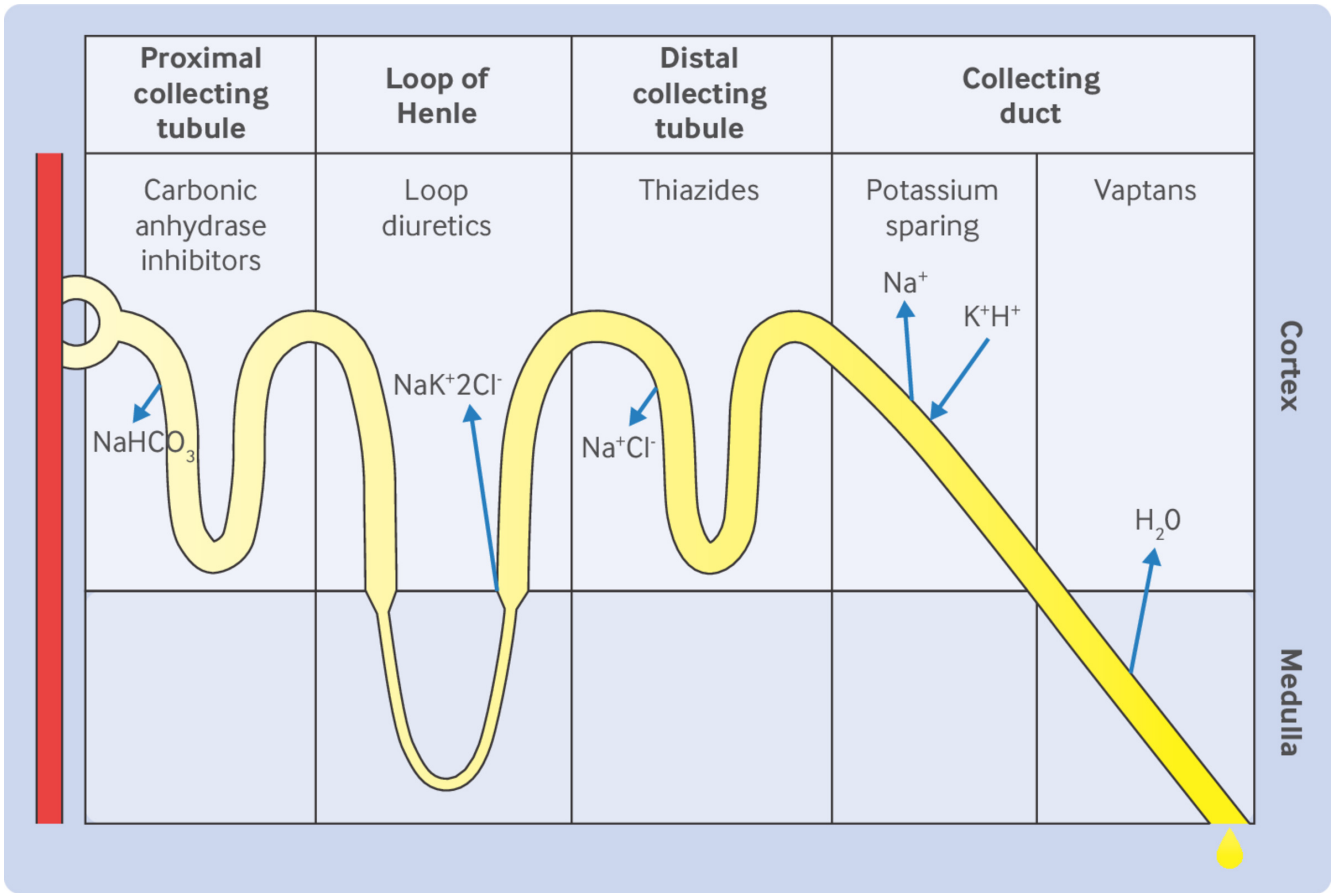

Fig 1 | Sites of action of common diuretics within the nephron. Each diuretic class inhibits sodium reabsorption at specific segments, disrupting electrolyte transport. For example, potassium sparing diuretics act on the late distal tubule and collecting duct, blocking sodium channels in principal cells. This reduces sodium reabsorption and decreases excretion of potassium and hydrogen ions, leading to increased sodium and water loss in the urine and conservation of potassium and hydrogen ions in the body.

There are two classes of potassium sparing diuretics. The first class is pure epithelial sodium channel blockers such as amiloride and triamterene. They cause mild diuresis by direct inhibition of sodium channels, which is helpful in managing hypertension, especially when used in combination with thiazide diuretics (such as hydrochlorothiazide/triamterene).

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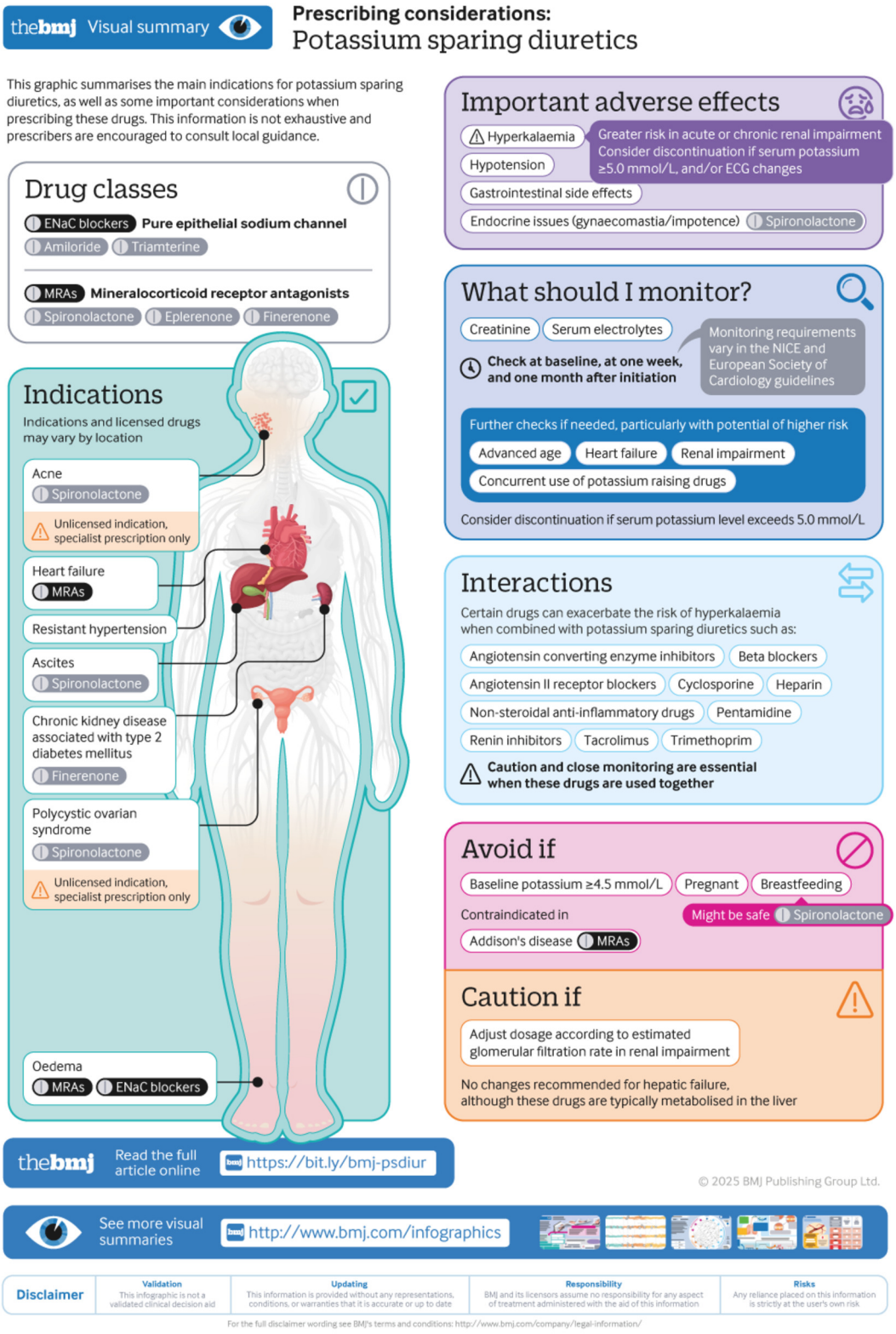
thebmj Visual summary
Prescribing considerations:
Potassium sparing diuretics
This graphic summarises the main indications for potassium sparing diuretics, as well as some important considerations when prescribing these drugs. This information is not exhaustive and prescribers are encouraged to consult local guidance.
Drug classes
ENaC blockers Pure epithelial sodium channel
Amiloride
Triamterine
MRAs Mineralocorticoid receptor antagonists
Spironolactone
Eplerenone
Finerenone
Indications
Indications and licensed drugs may vary by location
Acne
Spironolactone
Unlicensed indication, specialist prescription only
Heart failure
MRAs
Resistant hypertension
Ascites
Spironolactone
Chronic kidney disease associated with type 2 diabetes mellitus
Finerenone
Polycystic ovarian syndrome
Spironolactone
Unlicensed indication, specialist prescription only
Oedema
MRAs
ENaC blockers
Important adverse effects
Hyperkalaemia
Greater risk in acute or chronic renal impairment
Consider discontinuation if serum potassium ≥5.0 mmol/L, and/or ECG changes
Hypotension
Gastrointestinal side effects
Endocrine issues (gynaecomastia/impotence)
Spironolactone
What should I monitor?
Creatinine
Serum electrolytes
Check at baseline, at one week, and one month after initiation
Monitoring requirements vary in the NICE and European Society of Cardiology guidelines
Further checks if needed, particularly with potential of higher risk
Advanced age
Heart failure
Renal impairment
Concurrent use of potassium raising drugs
Consider discontinuation if serum potassium level exceeds 5.0 mmol/L
Interactions
Certain drugs can exacerbate the risk of hyperkalaemia when combined with potassium sparing diuretics such as:
Angiotensin converting enzyme inhibitors
Beta blockers
Angiotensin II receptor blockers
Cyclosporine
Heparin
Non-steroidal anti-inflammatory drugs
Pentamidine
Renin inhibitors
Tacrolimus
Trimethoprim
Caution and close monitoring are essential when these drugs are used together
Avoid if
Baseline potassium ≥4.5 mmol/L
Pregnant
Breastfeeding
Contraindicated in
Addison's disease
MRAs
Might be safe
Spironolactone
Caution if
Adjust dosage according to estimated glomerular filtration rate in renal impairment
No changes recommended for hepatic failure, although these drugs are typically metabolised in the liver
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The second class of potassium sparing diuretics is mineralocorticoid receptor antagonists such as spironolactone, eplerenone, and finerenone. They block sodium channels in the same way as pure epithelial sodium channel blockers,[2] but also suppress the renin-angiotensin-aldosterone system, providing additional therapeutic benefits (fig 2). By blocking aldosterone receptors, mineralocorticoid receptor antagonists not only reduce sodium reabsorption and conserve potassium but also counteract the maladaptive effects of chronic renin-angiotensin-aldosterone system activation. This article focuses on mineralocorticoid receptor antagonists because they are the most widely used potassium sparing diuretics in practice.

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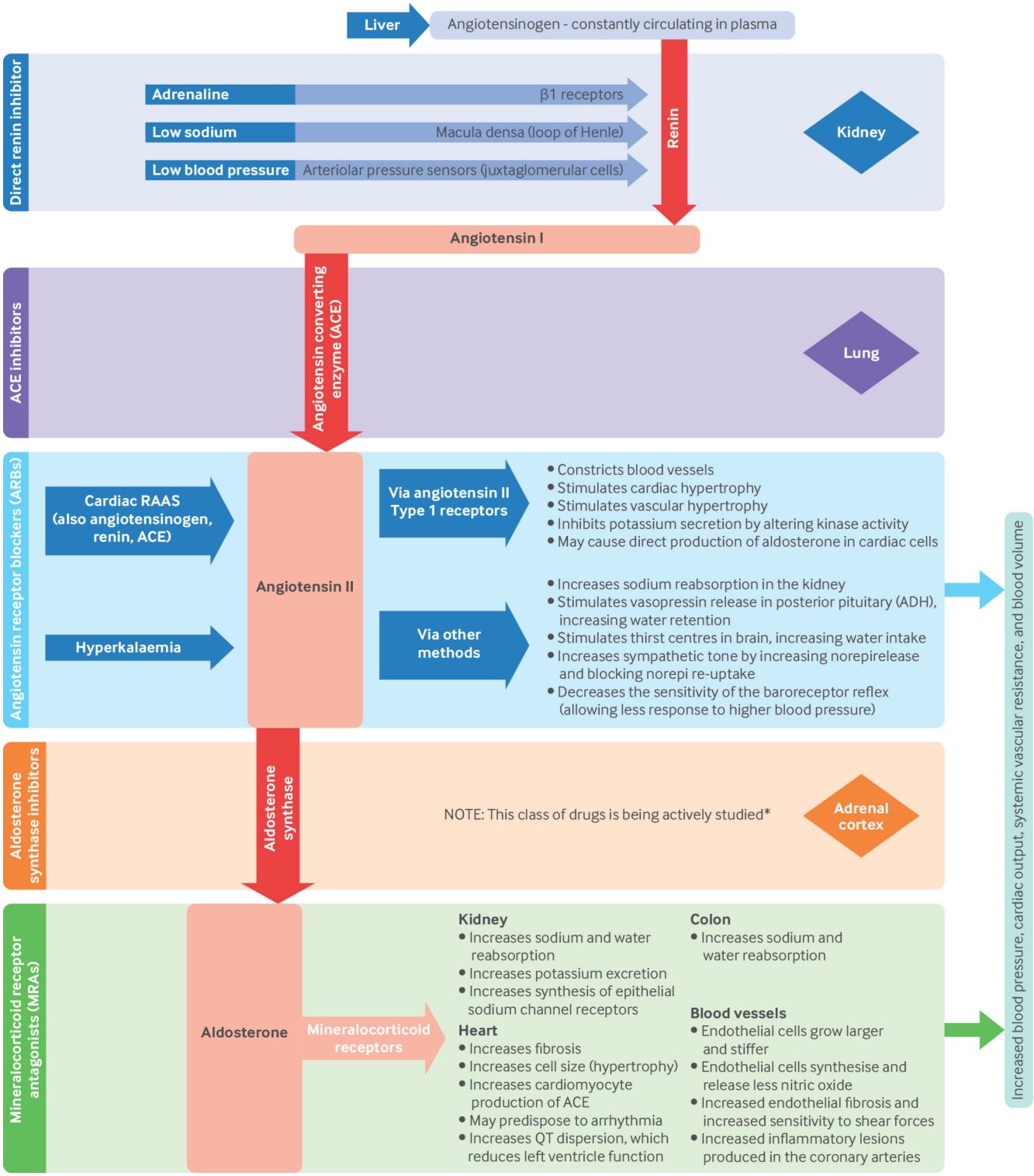
Liver
Angiotensinogen - constantly circulating in plasma
Direct renin inhibitor
Adrenaline
β1 receptors
Low sodium
Macula densa (loop of Henle)
Low blood pressure
Arteriolar pressure sensors (juxtaglomerular cells)
Renin
Kidney
Angiotensin I
ACE inhibitors
Angiotensin converting enzyme (ACE)
Lung
Angiotensin receptor blockers (ARBs)
Cardiac RAAS (also angiotensinogen, renin, ACE)
Hyperkalaemia
Angiotensin II
Via angiotensin II Type 1 receptors
Constricts blood vessels
Stimulates cardiac hypertrophy
Stimulates vascular hypertrophy
Inhibits potassium secretion by altering kinase activity
May cause direct production of aldosterone in cardiac cells
Via other methods
Increases sodium reabsorption in the kidney
Stimulates vasopressin release in posterior pituitary (ADH), increasing water retention
Stimulates thirst centres in brain, increasing water intake
Increases sympathetic tone by increasing norepirelease and blocking norepi re-uptake
Decreases the sensitivity of the baroreceptor reflex (allowing less response to higher blood pressure)
Aldosterone synthase inhibitors
Aldosterone synthase
NOTE: This class of drugs is being actively studied*
Adrenal cortex
Mineralocorticoid receptor antagonists (MRAs)
Aldosterone
Mineralocorticoid receptors
Kidney
Increases sodium and water reabsorption
Increases potassium excretion
Increases synthesis of epithelial sodium channel receptors
Heart
Increases fibrosis
Increases cell size (hypertrophy)
Increases cardiomyocyte production of ACE
May predispose to arrhythmia
Increases QT dispersion, which reduces left ventricle function
Colon
Increases sodium and water reabsorption
Blood vessels
Endothelial cells grow larger and stiffer
Endothelial cells synthesise and release less nitric oxide
Increased endothelial fibrosis and increased sensitivity to shear forces
Increased inflammatory lesions produced in the coronary arteries
Increased blood pressure, cardiac output, systemic vascular resistance, and blood volume

Fig 2 | The renin-angiotensin-aldosterone system (RAAS) illustrating key pathways, stimuli, and drug interventions. The RAAS is activated in response to conditions such as heart failure and other pathologies to help maintain blood pressure and fluid balance. However, prolonged or unchecked RAAS activation can lead to harmful effects on the heart and blood vessels, including hypertension, inflammation, and remodelling. This figure highlights selected RAAS components and corrective medications targeting these pathways. Adapted from Fels and Najjar.[3 4] *Current study[5]

Renin-angiotensin-aldosterone system activation can have short term physiological benefits, because it helps maintain blood pressure and kidney perfusion when renal blood flow is low. However, chronic activation becomes harmful, driving hypertension, cardiac remodelling, inflammation, and fibrosis. By increasing levels of angiotensin II and aldosterone, renin-angiotensin-aldosterone system activation narrows blood vessels, promotes sodium and water retention, raises blood pressure,

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and increases cardiac workload. Mineralocorticoid receptor antagonists prevent aldosterone from activating mineralocorticoid receptors, thereby reducing these downstream effects.[6]

Clinical uses

Spironolactone is the most frequently prescribed mineralocorticoid receptor antagonist, with approximately 340 000 prescriptions in the UK and 12.5 million in the US in 2022.[7 8]

Heart failure

Mineralocorticoid receptor antagonists are used to treat heart failure with reduced ejection fraction (defined as left ventricular ejection fraction <40%). Evidence supports a reduction in mortality,[9] and a reduction in the length of associated hospital stays.[10] Evidence is growing for use in heart failure with preserved ejection fraction (defined as left ventricular ejection fraction >50%), demonstrating that mineralocorticoid receptor antagonists reduce heart failure events and cardiovascular deaths,[11 12] regardless of the age of the patient.[13] These benefits appear to be additive in patients who are taking other drugs for heart failure, as directed by guidelines.[14] International guidelines that recommend the use of mineralocorticoid receptor antagonists in heart failure include National Institute for Health and Care Excellence,[15] American College of Cardiology/American Heart Association/Heart Failure Society of America,[16] and European Society of Cardiology.[17]

Eplerenone has identical dosing to spironolactone for this indication but is not associated with gynaecomastia or other endocrine related side effects seen with spironolactone. Some patients might prefer it for this reason. Eplerenone also demonstrated mortality and other outcome benefits in the EPHESUS (patients with myocardial infarction and low ejection fraction) and EMPHASIS-HF (mild or moderate heart failure and low ejection fraction) trials.[18 19]

The recent FINEARTS-HF trial showed that finerenone reduced worsening heart failure events and cardiovascular death, compared with placebo, in patients with heart failure with preserved ejection fraction and heart failure with mildly reduced ejection fraction (defined as left ventricular ejection fraction 40% to 50%).[20] This benefit is seen early in treatment,[21] particularly in patients with worsening disease,[22] without adversely affecting long term renal function,[23] and irrespective of age.[24] However, as yet, there are no head-to-head trials comparing the newer finerenone with well established spironolactone or eplerenone, suggesting that this may be a class effect.

Resistant hypertension

Resistant hypertension is defined as uncontrolled blood pressure despite three preferred agents such as a thiazide-type diuretic, a calcium channel blocker, and an ACE inhibitor or angiotensin II receptor blocker (ARB). The role of mineralocorticoid receptor antagonists in treating resistant hypertension is well established in multiple guidelines,[25] although this is an off-label indication in the UK. While potassium sparing diuretics provide only weak blood pressure reduction on their own, spironolactone and eplerenone are effective in resistant cases,[26] with studies showing clinically meaningful reductions in systolic blood pressure. Mineralocorticoid receptor antagonists have demonstrated reductions in systolic blood pressure of 9 mmHg compared with placebo in one study and 22/10 mmHg in another.[27 28] Resistant hypertension is considered a high risk condition, correlating with increased rates of diabetes mellitus, chronic kidney disease, ischaemic heart disease, and cerebrovascular disease.[29] More than 20% of patients with this condition have aldosterone excess hyperaldosteronism,[30] while many more are likely to have subclinical excess,[31] as this is often seen in overweight or obese patients.[32]

Renal effects

Because their diuretic potency is modest, potassium sparing diuretics are not usually used alone for oedema, but they can augment loop or thiazide diuretics by limiting sodium reabsorption later in the nephron. This combination can help overcome diuretic resistance in heart failure when further fluid removal is required. In nephrotic syndrome, potassium sparing diuretics combined with loop diuretics improve diuresis while mitigating potassium loss, though head-to-head comparisons between diuretic classes are lacking for this indication.[33] Finerenone provides a benefit for patients with chronic kidney disease,[34] including diabetic kidney disease.[35]

Ascites

Spironolactone has also shown greater efficacy in managing ascites in cirrhosis when compared with loop diuretics.[36 37] Although the trials were conducted using spironolactone and it is the most widely used clinically, this is likely to be a class effect. High levels of aldosterone lead to retention of sodium and water. By inhibiting aldosterone, mineralocorticoid receptor antagonists counteract this effect.[38]

Other indications

Spironolactone has been used to manage acne, with mechanisms of action involving multiple pathways,[39 40] including decreasing sebum excretion by 30% to 50%.[41] In addition, spironolactone has shown benefits in the treatment of polycystic ovarian syndrome owing to antiandrogen effects.[42]

What should I discuss with patients before starting treatment?

Begin by asking whether the patient has previously taken a potassium sparing diuretic, the reason it was prescribed, and how effective it was. Note that epithelial sodium channel blockers like amiloride and triamterene are often prescribed as part of combination pills, so patients might not always be aware that they have used these medications.

Explain that patients might not notice immediate changes, because it can take days to weeks for the full effect of the drug. Depending on the indication, benefits might include improvement in symptoms (such as reduced shortness of breath or swelling for patients with heart failure) or lowered blood pressure for patients with hypertension.

It is important to emphasise that even if symptoms do not noticeably improve, these drugs reduce risks such as hospital admissions for heart failure, and cardiovascular events including heart attacks and strokes.

NICE recommends that patients starting these drugs should be counselled about the increased risk of renal failure and hyperkalaemia in the context of dehydration, for example, secondary to an acute illness causing diarrhoea and vomiting. If patients develop diarrhoea and vomiting, they should consult their healthcare provider to consider stopping the treatment.[43]

What are important side effects to discuss?

Hyperkalaemia is an important side effect, although its incidence and severity are reported with high variability. The RALES trial reported a 2% incidence of hyperkalaemia in patients treated with spironolactone (12.5-25 mg daily) plus ACE inhibitors[44]; the

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ALBATROSS trial documented 3% (25 mg daily).[45] Other real world data have reported rates as high as 8%.[46] After the RALES trial was published, spironolactone use increased, which correlated with rises in hospital admissions related to hyperkalaemia from 2.4 to 11.0 per 1000 patients.[47] However, a subsequent analysis showed that with appropriate monitoring, the use of spironolactone still improved clinical outcomes, including mortality.[48] Notably, a UK study showed that between 1995 and 2007, hyperkalaemia incidence and hospital stays declined even as spironolactone use and potassium testing rose, likely owing to improved clinical vigilance.[49]

Fewer than 15% of hospital admissions for hyperkalaemia overall are attributed to potassium sparing diuretics, highlighting that while hyperkalaemia is important, it can be effectively managed with appropriate care.[50]

Some drugs (such as non-steroidal anti-inflammatory drugs, ACE inhibitors, ARBs, renin inhibitors, beta blockers, heparin, trimethoprim, pentamidine, cyclosporine, and tacrolimus) can exacerbate the risk of hyperkalaemia when combined with potassium sparing diuretics.[51] Caution and close monitoring are essential when these drugs are used together.

Despite the increased risk of hyperkalaemia from the combined use of potassium sparing diuretics and ACE inhibitors or ARBs,[52] this combination delivers the greatest mortality benefit in heart failure, especially in patients with reduced ejection fraction.[53]

The risk of hyperkalaemia is greater in patients with acute or chronic renal impairment,[54] and guidelines often recommend not initiating treatment if baseline potassium exceeds 4.5 mmol/L, with discontinuation advised if levels surpass 5.0 mmol/L.[55]

However, in the RALES trial, potassium levels were higher in the spironolactone group than the placebo group, and the mortality advantage of spironolactone persisted up to potassium levels of 5.5 mmol/L, suggesting that modest levels of hyperkalaemia in this setting might not be disadvantageous.[56] Additionally, there is some evidence that indicates that spironolactone might be well tolerated in patients with high potassium levels and renal failure.[57]

Steroidal mineralocorticoid receptor antagonists (eg, spironolactone) additionally interact with androgen and progesterone receptors, with the potential to cause endocrine side effects such as gynaecomastia,[58] impotence, and menstrual irregularities. Gynaecomastia caused by spironolactone is related to the dose, affecting about 9% of patients taking the recommended dose (25 mg daily) and up to 52% at high doses (150 mg daily), and is generally reversible after discontinuation.[59] Hormonal side effects are substantially less common with eplerenone and particularly rare with non-steroidal agents such as finerenone,[60] which are more selective for mineralocorticoid receptors and carry a lower risk of hyperkalaemia and hormonal effects. Longer acting diuretic formulations might increase nocturia, but potassium sparing diuretics typically cause less urinary frequency and urgency than loop or thiazide diuretics,[61 62] owing to their lower diuretic potency.

Other side effects shared by antihypertensive or diuretic classes include hypotension (including orthostatic hypotension),[63] erectile dysfunction (thiazides, beta blockers, and spironolactone),[64] and gastrointestinal symptoms, including nausea, diarrhoea, and constipation, although these tend to be mild and short lived. Note that nausea and vomiting can also be caused by hyperkalaemia, so electrolytes should be checked if symptoms occur. Hyponatraemia can occur with all diuretics but appears less common with potassium sparing agents, potentially owing to their weaker diuretic effect.[65]

In practice, awareness of these side effects, particularly hyperkalaemia and hormonal effects, alongside diligent monitoring, enables patients and clinicians to balance risks and benefits effectively, optimising patient safety and clinical outcomes.

This list of side effects is not exhaustive, and we encourage prescribers to refer to local guidelines.

What to consider when prescribing?

Potassium sparing diuretics are generally avoided in pregnancy, but spironolactone is considered acceptable for use during breastfeeding in several settings, including the UK,[66] despite a small amount of transfer into breast milk. There are no breastfeeding data for eplerenone, and finerenone is contraindicated in breastfeeding. Mineralocorticoid receptor antagonists are contraindicated in Addison's disease, a condition already marked by aldosterone deficiency.[67]

Potassium sparing diuretics are unique among diuretics, in that their effect is based on serum concentrations, not on drug concentrations inside the nephron ("intraluminal"). For this reason, creatinine clearance is not a primary factor in determining dosing with potassium sparing diuretics, as it would be in other diuretics, which do need to be passed into the nephron to be effective. Additionally, they are unique among diuretics (other than ethacrynic acid) in not containing a sulfa moiety.[68] They are also unique in the fact that unlike most other diuretic classes, potassium sparing diuretics are not associated with causing gout.[69]

Table 1 outlines potassium diuretics and their characteristics. Canrenone was not included in the table owing to its low clinical use.

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Table 1 | Dosing recommendations for potassium sparing diuretics listed by class (lower or higher doses may be used based on clinical discretion)

Dose	Duration (h)	Peak effect (h)	Renal dosing
Mineralocorticoid receptor antagonists			
Spironolactone (Steroidal. Most potent and most likely drug to cause hyperkalaemia and/or gynecomastia)[70]			
Heart failure: start 25 mg, increase to 50 mg or higher Hypertension: start 25 mg daily, can increase to 100 mg daily*[71]	48-72	2-4	GFR >50: no change GFR 30-50: 12.5 mg daily or every other day; double as tolerated to max 25 mg daily (if K+ <5 and renal function stable) GFR <30: not recommended
Eplerenone (Steroidal)[72]			
Heart failure: start 25 mg, target 50 mg daily Hypertension: start 25 mg, can increase to 50 mg twice daily	Steady state in 48	1.5-2 (full effect in 4 weeks)	GFR >50: no change GFR 30-50: 12.5 mg daily or every other day; double as tolerated to max 25 mg daily (if K+ <5 and renal function stable) GFR <30: not recommended
Finerenone (Non-steroidal)[68]			
Heart failure: GFR >60: 20 mg daily GFR 25-60: 10 mg daily GFR <25: not recommended	Steady state in 48	0.5-1.25	Adjust maintenance dose based on serum potassium levels
Epithelial sodium channel blockers			
Amiloride[73]			
Oedema or hypertension: 5 mg daily, can increase to 5 mg twice daily or 10 mg daily	~24	3-4	CrCl 10-50: 1/2 dose CrCl <10: avoid CrCl <30 and aged >65: avoid
Triamterene†[74]			
Oedema: 50-100 mg daily, increase to max 300 mg daily in divided doses	7-9 of diuresis	2-4 for diuretic effect (might take several days for optimal response)	CrCl <50: avoid CrCl <30 and aged >65: avoid

* This differs from the British National Formulary. †Not available as monotherapy in the UK, only in combination with other agents. GFR=glomerular filtration rate, in mL/min/1.73 m2; CrCl=creatinine clearance, in mL/min

What should I monitor during the course of the prescription?

Serum electrolytes should be monitored when taking any diuretic drug, but special attention should be paid when taking potassium sparing diuretics with other drugs that can increase potassium. Many American guidelines recommend checking serum electrolytes and creatinine at baseline, at one week, and one month after initiation of these diuretic drugs because hyperkalaemia typically occurs within four weeks of initiation.[16] All current guidelines also recommend longitudinal lab work at least every six months, or if there is reason for clinical concern, particularly concerns regarding renal function. There are some additional variations in frequency of monitoring between the NICE and European Society of Cardiology guidelines.[75-77]

Potassium supplementation can be given to patients who experience hypokalaemia while taking loop diuretics, although supplementation may no longer be necessary when potassium sparing diuretics are added. The British National Formulary states that potassium supplementation should be avoided with potassium sparing diuretics, however, we recognise in our own practice that this is sometimes required to treat clinically important hypokalaemia, in which case we would suggest additional monitoring.

Although possible with any diuretic medication, hyponatraemia appears to be less common with potassium sparing diuretics—most severe cases of hyponatraemia are associated with thiazides.[78]

What other concerns might my patient have?

In our experience, patient concerns tend to focus on urine output, low blood pressure, and the need for monitoring of serum electrolytes. However, the risks of these effects are low with potassium sparing diuretics. In general, increased diuresis tends to abate in many patients after one to two weeks, and the diuretic effect with this class is weak enough that it might not be noticed at all. Blood pressure effects persist after the diuretic effect has waned. Often, blood pressure lowering is an intended outcome. Concerns about low blood pressure can be addressed by initiating home blood pressure or ambulatory blood pressure monitoring.

When should I stop the prescription?

These drugs are used for the duration of the underlying condition; in most cases, this would be lifelong. However, it is reasonable to consider discontinuation when the drug is no longer needed, for example, for control of hypertension or oedema, if there are adverse effects, or for end-of-life care.

Hyperkalaemia is a reason to stop potassium sparing diuretics. There is no clear consensus regarding a threshold value that should prompt discontinuation, but a pragmatic approach would be to stop them if there is persistent hyperkalaemia that causes electrocardiogram changes or prompts hospital admission. Some guidelines recommend discontinuation on a 30% decrease in estimated glomerular filtration rate, although those same guidelines also recommend continuing the combination of ACE I and/or ARB in patients with chronic kidney disease, even if the estimated glomerular filtration rate falls below 30 mL/min per 1.73 m^2.[79] Note that some degree of increase in potassium is expected, as is a decrease in glomerular filtration rate, and an increase in creatinine. These result from direct drug effects, as well as indirect effects of the intended relative hypovolaemia with effective diuresis. In the SOGALDI-PEF trial, one in five patients had a drop of >30% in estimated glomerular filtration rate when combining

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mineralocorticoid receptor antagonists and sodium-glucose co-transporter 2 inhibitors, although this combination yielded noticeable benefits.[80] Stopping medication might undermine the benefits of this combination therapy, a particular problem given that these medications are subsequently often not restarted.[81]

Summary of product characteristics

Mechanism of action

- All block reabsorption of sodium and water in the collecting duct, increasing urine production and salt wasting
- Mineralocorticoid receptor antagonists also inhibit aldosterone receptors renally and systemically

Indications

- Hypertension
- Oedema
- Heart failure (spironolactone, eplerenone, finerenone)
- Renal failure from type 2 diabetes (finerenone)
- Ascites secondary to cirrhosis

Common adverse events

- Gynaecomastia
- Hyperkalaemia
- Gastrointestinal effects, such as nausea, vomiting, constipation

Interactions

- These drugs can interact with hundreds of other drugs, particularly drugs that increase potassium levels or affect renal function (eg, angiotensin converting enzyme inhibitors/ angiotensin II receptor blockers, various antibiotics and antifungals, and non-steroidal anti-inflammatory drugs)

Cautions

- Drug interactions
- Hyperkalaemia
- Renal impairment

Prescribing in renal/hepatic impairment

- No changes recommended for hepatic failure, although these drugs are typically metabolised in the liver
- Dosing adjustments are required for various degrees of renal failure because these drugs are excreted by the kidneys

Pregnancy and breastfeeding

- These drugs are not labelled as safe for use in pregnancy. Spironolactone might be safe during breastfeeding, others might not

Table of equivalence

- For blood pressure effect, 1 mg of spironolactone is equivalent to 4.5 mg of eplerenone, and 3.3 mg of amiloride[82]

How patients were involved in the creation of this article

No patients were involved in the creation of this article.

Education into practice

- In what clinical situations might you consider a potassium sparing diuretic for your hypertensive patients?
- How do you decide between potassium sparing diuretics and other diuretics for oedema? If you prescribe a potassium sparing diuretic, how do you select between the various options?

Further information resources

- European Society of Cardiology/European Society of Hypertension guidelines[83]
- NICE guidelines for the diagnosis and management of hypertension[84]
- American College of Cardiology/American Heart Association consensus guidelines[85]

Competing interests: *The BMJ* has judged that there are no disqualifying financial ties to commercial companies. Further details of The BMJ policy on financial interests is here: https://www.bmj.com/sites/default/files/attachments/resources/2016/03/16-current-bmj-education-coi-form.pdf.

Patient consent: The patient vignette was a composite of various patients and does not reflect any identifiable facts regarding a particular patient.

Provenance and peer review: Commissioned, externally peer reviewed.

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